Committee on Environmental Hazards
Committee on Accident and Poison Prevention

Statement on Childhood Lead Poisoning

Lead remains a significant hazard to the health of American children.[1-3] Virtually all children in the United States are exposed to lead that has been dispersed in air, dust, and soil by the combustion of leaded gasoline. Several hundred thousand children, most of them living in older houses, are at risk of ingesting lead-based paint as well as lead-bearing soil and house dust contaminated by the deterioration of lead-based paint. Although the incidence of symptomatic lead poisoning and of lead-related mortality has declined dramatically,[4] data from targeted screening programs[1] and from a national survey[2] show that there are many asymptomatic children with increased absorption of lead in all regions of the United States. It is particularly prevalent in areas of urban poverty.

Childhood lead poisoning can readily be detected by simple and inexpensive screening techniques; however, screening is sporadic and in some areas not available.

Despite wide recognition of the importance of preventing children's exposure to lead, state and federal funding for the screening of children and for the removal of environmental lead hazards has diminished in recent years. Thus, pediatricians attempting to address the problem of childhood lead exposure face serious economic and administrative obstacles to effective intervention.

This statement reviews current approaches to the diagnosis, treatment, and prevention of lead poisoning, and it recommends steps to reduce the pervasive impact of lead on children's health. Some of these recommendations are addressed to practitioners and others to agencies of state and federal government. It is important to recognize that virtually all of these preventive steps are after the fact. Ideally, in keeping with the precepts of primary prevention, lead should have been prohibited from ever having become dispersed in the modern environment.

PEDIATRICS (ISSN 0031 4005).

BACKGROUND AND DEFINITIONS

Lead has no biologic value. Thus, the ideal whole blood lead level is 0 μg/dL. According to the Second National Health and Nutrition Examination Survey (NHANES II), conducted from 1976 to 1980,[2] the mean blood lead level in American preschool children was approximately 16 μg/dL. Substantially lower lead levels are seen in persons remote from modern industrialized civilization[5] and in the remains of prehistoric man.[6]

Until recently, whole blood lead levels as high as 30 μg/dL were considered acceptable. However, disturbances in biochemical function are demonstrable at concentrations well below that figure. For example, inhibition of δ-aminolevulinic acid dehydrase, an enzyme important to the synthesis of heme, occurs at whole blood lead levels below 10 μg/dL.[7,8] Also, the enzyme ferrochelatase, which converts protoporphyrin to heme, is inhibited in children at a blood lead concentration of approximately 15 μg/dL; thus, elevations in erythrocyte protoporphyrin above normal background become evident at blood lead levels above 15 μg/dL.[9] In addition, depression of circulating levels of 1,25-dihydroxyvitamin D (the active form of vitamin D) is seen at blood lead levels well below 25 μg/dL.[10,11]

Neuropsychologic dysfunction, characterized by reduction in intelligence and alteration in behavior, has been shown conclusively to occur in asymptomatic children with elevated blood lead levels.[12-15] The results of clinical and epidemiologic studies conducted in the United States,[12] Germany,[13] and England[14] indicate clearly that blood lead levels below 50 μg/dL cause neuropsychologic deficits in asymptomatic children. Recent clinical and experimental studies suggest that neuropsychologic damage may be produced in children with blood lead levels below 35 μg/dL.[15]

Short stature, decreased weight, and diminished chest circumference have recently been found in analyses of data from the NHANES II survey to be significantly associated with blood lead levels in American children younger than 7 years of age,

after controlling for age, race, sex, and nutritional status. Although the effects are small, the results are statistically robust.[16]

In light of these data, an expert Advisory Committee to the Centers for Disease Control (CDC) has determined that a blood lead level of 25 μg/dL or above indicates excessive lead absorption in children and constitutes grounds for intervention.[17] Increased lead absorption was previously defined by a blood lead level of 30 μg/dL. Furthermore, the CDC committee has now defined childhood lead poisoning as a blood lead level of 25 μg/dL in association with an erythrocyte protoporphyrin level of 35 μg/dL or more.[16] The Academy concurs in these definitions. Also, the Academy anticipates that as evidence of the low-dose toxicity of lead continues to develop, these definitions will be lowered still further.

PREVALENCE OF LEAD POISONING

Data from NHANES II[2] indicate that between 1976 and 1980 the national prevalence of blood lead levels of 30 μg/dL or higher was 4% among American children 6 months to 5 years of age. Applying this rate to US census data, it may be estimated that, between 1976 and 1980, 780,000 American preschool children had excess levels of lead in their blood. In the NHANES II data, there was wide disparity in the prevalence of elevated blood lead levels between black children (12%) and white children (2.0%) irrespective of social class or place of residence. A similar disparity was noted in mean blood lead levels, which were 21 μg/dL in black preschool-aged children and 15 μg/dL in white children of the same age. Prevalence rates for elevated blood lead levels were highest among families in densely populated urban areas and in those with incomes of less than $15,000 per year. However, it should be noted that cases of lead poisonings were found also in families of higher income and in rural settings.

Between 1976 and 1980, the average blood lead level in Americans of all ages decreased from 15.8 to 10.0 μg/dL according to the NHANES II.[4] This decrease coincided with a reduction in the use of lead additives in gasoline. Additional factors in this reduction may have included a simultaneous reduction in the lead content of foodstuffs, the impact of targeted screening programs in high-risk areas, and an increase in public awareness of the hazards of lead.

SOURCES OF LEAD

Environmental Sources

Lead is ubiquitous. A natural constituent of the earth's crust, lead may be found in drinking water, soil, and vegetation. Its low melting point, malleability, and high density, as well as its ability to form alloys, have made lead useful for myriad purposes. Many of these uses (eg, radiation shields, storage batteries) are not intrinsically dangerous. However, when lead is used for purposes other than intended (eg, burning of storage battery casings), when it is incorrectly applied or removed (eg, improper use of lead ceramic glazes, burning and sanding of old leaded paint), when it is disseminated rather than reused (combustion of lead additives in automotive fuels), or when it is improperly discarded, lead enters the human environment in potentially hazardous form.

For purposes of estimating risk to children, lead sources may be categorized as low, intermediate, and high dose (Table 1).

Low-Dose Sources. These sources of lead include air, food, and drinking water. Together, these sources, which have accounted for an average estimated blood lead concentration of approximately 10 μg/dL in the recent past, probably now account for a blood level concentration of about 6 μg/dL. Mean ambient air lead concentrations currently average less than 1 $\mu g/m^3$ of air, although in areas near lead smelters, concentrations may be substantially higher.[18]

Average dietary intake of lead increases from 20 μg/d during early infancy to 60 to 80 μg/d by 5 to 6 years of age.[19] Except in isolated areas, it would appear that the majority of public drinking water supplies in the United States have a lead concentration of less than 20 μg/dL. However, these data may be misleading if, as is generally the case, the water samples have been obtained from the distribution plant prior to the distribution of water through a plumbing system that contains lead pipes. The lead solvency of drinking water can be reduced by reducing acidity of water supplies and by abandoning the use of lead-based solder at pipe joints in new and replacement plumbing. Communities with excessive lead in water have successfully used each of these remedial approaches.

TABLE 1. Common Sources of Lead

Low dose
Food
Ambient air
Drinking water
Intermediate dose
Dust (household)
Interior paint removal
Soil contaminated by automobile accident
Industrial sources
Improper removal of exterior paint
High dose
Interior and exterior paint

Intermediate-Dose Sources. These sources include dust and soil in children's play areas. Dust and soil are contaminated principally by automotive exhaust and by the weathering and deterioration of old lead paint (both interior and exterior). Although background soil lead contaminants in rural areas are generally less than 200 ppm, concentrations of lead in urban soil can exceed 3,000 ppm. In industrial areas where lead smelters have been situated (eg, El Paso, TX; Kellogg, ID), the lead content of dust can, however, exceed 100,000 ppm,[18] thus producing significant elevations in children's blood lead levels. Each increase of 100 ppm in the lead content of surface soil above a level of 500 ppm is associated with a mean increase in children's whole blood lead levels of 1 to 2 μg/dL. When dust and soil are the only sources of exposure to lead, symptoms are rarely encountered, although lead toxicity may occur. Soil lead may, however, be extremely difficult to abate, and chronic low-grade ingestion may continue undetected even after a child has come to medical attention. The proper site for disposal of lead waste, such as lead-contaminated soil, is a hazardous waste facility that has been approved by the US Environmental Protection Agency.

High-Dose Sources. These sources are those in which the concentrations of lead are sufficient to produce acute and potentially fatal illness. Lead-based paint on both the interior and exterior surfaces of housing remains the most common high-dose source of lead for preschool-aged children. It continues to be the experience of most pediatricians that virtually all cases of symptomatic lead poisoning and blood lead levels greater than 70 μg/dL result from the ingestion of lead paint chips.

Lead-based paint is still widespread. A 1978 US census survey found that 8 million of the 27 million occupied dwellings in the United States, which had been built prior to 1940 when use of lead-based paint was common,[20] were deteriorated or dilapidated. An additional 22 million dwellings were built between 1940 and 1960, and 75% of these units are estimated to contain lead-based paint. Nationally, according to the 1978 census survey of housing, 9% of rental units have peeling paint.[21]

Although the use and manufacture of interior lead-based paint declined dramatically during the 1950s, exterior lead-based paint continued to be available until the mid-1970s and is still available for maritime use, farm and outdoor equipment, road stripes, and other special purposes. Thus, potential for domestic misuse of lead-based paint continues to exist. Manufacturers could voluntarily decrease the lead content of interior paint until 1977, when the US Consumer Product Safety Commission enacted regulations banning the sale in interstate commerce of paints for exposed interior and exterior residential surfaces containing more than 0.06% lead by weight in final, dry solid form.

A previously unforeseen, but increasingly recognized, danger is that of improper removal of lead-based paint from older houses during renovation or, ironically, during cleaning to protect children. Torches, heat guns, and sanding machines are particularly dangerous because they can create a lead fume.[22] Sanding not only distributes lead as a fine dust throughout the house but also creates small particles that are more readily absorbed than paint chips. The greatest hazard in paint removal appears to be to the person doing the "deleading" and to the youngest children in the dwelling. There may be significant morbidity. Persons who perform this work should comply with the standards for occupational exposure to lead which have been developed by the US Occupational Safety and Health Administration. Pregnant women, infants, and children should be removed from the house until deleading is completed and cleanup accomplished. Proper cleaning of the dust and chips produced in deleading must include complete removal of all chipping and peeling paint and vacuuming and thorough wet mopping, preferably with high-phosphate detergents. This waste must be discarded in a secure site.

Another previously unrecognized hazard lies in sandblasting. This technique is commonly used to remove lead from exterior surfaces. There are no standardized safeguards. Recent case reports of lead poisoning among sandblasters underscore the hazard.[23] Sandblasting creates large amounts of lead-laden dust and debris which, if improperly disposed or not properly removed, redouble the hazard.

Uncommon Sources (Table 2)

Additional lead sources include hobbies such as artwork with stained glass and ceramics, particularly when conducted in the home. Folk medicines

TABLE 2. Uncommon Sources of Lead

Metallic objects (shot, fishing weight)
Lead glazed ceramics
Old toys and furniture
Storage battery casings
Gasoline sniffing
Lead plumbing
Exposed lead solder in cans
Imported canned foods and toys
Folk medicines (eg, azarcon, Greta)
Leaded glass artwork
Cosmetics
Antique pewter
Farm equipment

used to treat gastrointestinal ailments may contain lead and mercuric or arsenical salts. Recent reports have noted lead poisoning from use of azarcon (lead tetroxide)[24] and Greta (lead monoxide) among Mexican-Americans and from use of Pay-loo-ah, a Chinese folk remedy,[25] among Hmong refugee children. Cosmetics (ceruse, surma, or kohl), particularly those from Asia, may contain white lead or lead sulfide[26,27] and have caused severe lead poisoning. Another source of lead is improperly soldered cans, particularly those containing acidic foodstuffs. Food should not be heated in such cans, as heating increases the dissolution of lead. Pediatricians should realize as a practical matter that the lead content of imported earthenware toys, medicines, or canned foods cannot readily be regulated. In addition, antique toys, cribs, and utensils may have a significant lead content.

Lead-glazed pottery is a potential source of lead in food and drink. If not fired at high temperatures, lead may be released from the glaze in large amounts when such pottery is used for cooking or for storage of acidic foodstuffs. Also, if pottery vessels are washed frequently, even a properly fired glaze can deteriorate, releasing unsafe levels of previously adherent lead.[28] Sporadic cases of plumbism have been traced to lead-glazed pottery.[29]

Among the oldest sources of lead in America is antique pewter. Food should not be cooked or stored in antique pewter vessels or dishes. Although uncommon, many of the above sources have been associated with severe, symptomatic, and even fatal lead poisoning.

Finally, a number of cases of lead poisoning have been reported among the children of workers in smelters, foundries, battery factories, and other lead-related industries.[30,31] These workers can bring home highly concentrated lead dust on their skin, shoes, clothing, and automobiles. This source of exposure can be avoided by providing showers at work, by providing workers with a change of clothing, and by having clothing laundered at the workplace.

In summary, it can be inferred from the NHANES II data that most children in the United States with increased lead absorption have been exposed to low-dose or to intermediate-dose lead sources. Four percent of children have blood lead levels in excess of 30 μg/dL, but only 0.1% have levels exceeding 50 μg/dL.[2]

ROUTES OF ABSORPTION

Ingestion is the principal route of lead absorption in children. Because of the high density of lead, ingestion of surprisingly small quantities may produce toxic effects. A lead paint chip weighing only 1 g (approximately 1 cm^2 in surface area) and containing 5% lead by weight will deliver a potential dose of 50 mg (50,000 μg); by comparison, the safe upper limit for daily intake of lead by children is 5 μg/kg of body weight.[32] Because ingestion of such chips is not uncommon, it might be expected that large numbers of children would have symptomatic lead poisoning. However, most ingested chips are swallowed whole or in large pieces, rendering much of the lead contained in them unavailable for absorption.

Several recent studies have reported the effectiveness of normal hand-to-mouth activity as a means for the transfer of lead-laden dust from the environment into children.[33–35] True pica (the indiscriminate ingestion of nonfood substances), although still an important risk factor,[36] need not be present.

Inhalation is the second major route of lead absorption for children. Lead absorbed by way of the lungs contributes in additive fashion to the total body lead burden. The efficiency of respiratory absorption depends on the diameter of airborne lead particles. For most common lead aerosols of mixed particle size, it has been estimated that between 30% and 50% of total inhaled lead is contained in particles of sufficiently small diameter (less than 5 μm) to be retained in the lungs and absorbed. Larger particles deposit in the nose, throat, and upper airways where they are cleared by ciliary action and then are either expectorated or swallowed.

PREDISPOSING FACTORS

Factors known to increase susceptibility to lead toxicity include nutritional deficiencies and age-related oral behavior (with or without pica) (Table 3).

Animal and human studies[37] have shown that deficiencies of iron, calcium, and zinc all result in increased gastrointestinal absorption of lead. Of particular concern is the effect of lack of iron, because the prevalence of iron deficiency in infancy is at least 15% and may be higher.[38] Iron deficiency, even in the absence of anemia, appears to be the single most important predisposing factor for increased absorption of lead. Conspicuous examples of nutritional iron deficiency include breast-fed infants and "milk babies" who may receive little food

TABLE 3. Predisposing Factors for Lead Poisoning

Nutritional deficiency of iron, calcium, or zinc
Sickle cell disease
Young age
Hand-to-mouth activity, including pica
Metabolic disease

other than milk until 12 to 18 months of age. In the presence of iron deficiency insufficient to produce anemia, gastrointestinal absorption of lead is increased severalfold.

SCREENING

Screening for lead poisoning is sporadic. Methods used have included determination of blood lead level, erythrocyte protoporphyrin level, or both.

Current recommendations from the CDC call for annual or semiannual screening of children in high-risk settings or with significant predisposing factors.[17] To guide the interpretation of screening results, the CDC has developed a series of guidelines (Tables 4 and 5).

The erythrocyte protoporphyrin determination provides a sensitive and inexpensive screen for both increased lead absorption and iron deficiency, two of the most common preventable health problems in childhood[38]; elevation in the erythrocyte protoporphyrin level can reflect iron deficiency before anemia becomes clinically evident. There is increasing interest, therefore, in adopting the erythrocyte protoporphyrin determination as a screening tool for both problems, particularly because it is more sensitive to iron deficiency than the hematocrit.[39]

Both capillary tubes and filter paper have been used for obtaining screening samples. Capillary tubes are cumbersome but have the advantage of providing sufficient blood for concomitant lead determination if the erythrocyte protoporphyrin level is elevated. Filter paper sampling provides ease of collection and transport, but the accuracy of analyses based on filter paper samples is not yet established. Determination of the blood lead level by fingerstick sampling is subject to contamination by lead on the skin, whether collection is by capillary tube or filter paper. Such contamination does not affect the determination of the erythrocyte protoporphyrin level.

Two analytical techniques are available for determination of erythrocyte protoporphyrin: (1) extraction of protoporphyrin from erythrocytes and subsequent measurement in a fluorimeter and (2) direct fluorimetry of a thin layer of RBCs (hematofluorometer). Because values derived from these two methods may differ (Tables 4 and 5), a pediatrician should be aware of which is in use. When in doubt, the extraction method is preferred, because of its greater reproducibility, particularly at lower concentrations of erythrocyte protoporphyrin.

TABLE 4. Zinc Protoporphyrin by Hematofluorometer: Risk Classification of Asymptomatic Children for Priority Medical Evaluation*

Blood Lead (μg/dL)	Erythrocyte Protoporphyrin (μg/dL)			
	<35	35–74	75–174	>175
Not done	I	†	†	†
<24	I	Ia	Ia	‡
25–49	Ib	II	III	III
50–69	§	III	III	IV
>70	§	§	IV	IV

* Diagnostic evaluation is more urgent than the classification indicates for (1) children with any symptoms compatible with lead toxicity, (2) children younger than 36 months of age, (3) children whose blood lead and erythrocyte protoporphyrin levels place them in the upper part of a particular class, (4) children whose siblings are in a higher class. These guidelines refer to the interpretation of screening results, but the final diagnosis and disposition rest on a more complete medical and laboratory examination of the child.
† Blood lead test needed to estimate risk.
‡ Erythropoietic protoporphyria. Iron deficiency may cause elevated erythrocyte protoporphyrin levels up to 300 μg/dL, but this is rare.
§ In practice, this combination of results is not generally observed; if it is observed, immediately retest with whole blood.

TABLE 5. Erythrocyte Protoporphyrin (EP) by Extraction: Risk Classification of Asymptomatic Children for Priority Medical Evaluation*

Blood Lead (μg/dL)	EP (μg/dL)			
	<35	35–109	110–249	>250
Not done	I	†	†	†
<24	I	Ia	Ia	‡
25–49	Ib	II	III	III
50–69	§	III	III	IV
>70	§	§	IV	IV

* Diagnostic evaluation is more urgent than the classification indicates for (1) children with any symptoms compatible with lead toxicity, (2) children younger than 36 months of age, (3) children whose blood lead and EP levels place them in the upper part of a particular class, (4) children whose siblings are in a higher class. These guidelines refer to the interpretation of screening results, but the final diagnosis and disposition rest on a more complete medical and laboratory examination of the child. Screening tests are not diagnostic. Therefore, every child with a positive screening test result should be referred to a physician for evaluation, with the degree of urgency indicated by the risk classification. At the first diagnostic evaluation, if the screening test was done on capillary blood, a venous blood lead level should be determined in a laboratory that participates in the Centers for Disease Control's blood lead proficiency-testing program. Even when tests are done by experienced personnel, blood lead levels may vary 10% to 15%, depending on the level being tested. Tests for the same child may vary as much as ±5 μg/dL in a 24-hour period. Thus, direction should not necessarily be interpreted as indicative of actual changes in the child's lead absorption or excretion.
† Blood lead test needed to estimate risk.
‡ Erythropoietic protoporphyria. Iron deficiency may cause elevated EP levels up to 300 μg/dL, but this is rare.
§ In practice, this combination of results is not generally observed; if it is observed, immediately retest with whole blood.

It is most important to note that screening tests are not diagnostic. Every child with a positive screening test result should be referred to a pediatrician for further evaluation, with the urgency of referral indicated by the risk classification (Tables 4 and 5). At the first diagnostic evaluation, if the screening test was performed on capillary blood, a venous blood lead level should be determined in a laboratory that participates in an accredited blood lead proficiency-testing program. To reduce the likelihood of false-positive results, lead-free syringes, needles, and tubes must be used in obtaining venous blood samples for lead analysis.

The developmentally disabled who reside in "halfway houses" or community residences or who attend school in older buildings deserve special attention in lead-screening programs. Because this population may be older than preschool age, protective statutes may not recognize their high-risk status, particularly with respect to pica behaviors. Physicians caring for developmentally disabled patients should be aware that their risk of lead ingestion may continue long beyond the age of 6 years.

INTERVENTION

Once a diagnosis of increased lead absorption has been confirmed by venous blood lead determination, the sine qua non of intervention is the prompt and complete termination of any further exposure to lead.[40] This intervention requires accurate identification of the source of lead and either its removal or removal of the child from the unsafe environment. Some states (eg, Massachusetts) have passed stringent legislation requiring prompt removal of lead hazards in cases of lead poisoning, and there are strong penalties for failure to comply. *At all costs, a child should not be permitted to enter or to be present in a leaded environment during deleading until the deleading, subsequent cleanup, and reinspection have been satisfactorily completed.* Although some regulations call only for removal of leaded paint from "chewable" surfaces (eg, window sills and door frames) or up to a height of 1.2 m (4 ft), all chipping and peeling paint should be removed from all surfaces, particularly from ceilings.

After deleading, the house must be thoroughly cleaned and reinspected to assure compliance with safety regulations. Indeed, repeated thorough cleaning is advisable, especially in the case of deteriorated or dilapidated housing. High-phosphate detergents are particularly useful in removing lead dust. Children should not return home until cleaning is completed.

Medical intervention should begin with throrough clinical evaluation including diagnostic studies of lead toxicity and, when indicated, a lead mobilization test.[40] Diagnostic studies should include a blood cell count with RBC indices, a reticulocyte count, and, if indicated and available, tests of serum iron and iron-binding capacity, and a serum ferritin assay. Routine urinalysis might be considered. Because chelating agents are potentially nephrotoxic, BUN and/or serum creatinine values should be determined before chelation to rule out occult renal disease either secondary to plumbism or preexisting.[41] Roentgenographic studies to be considered include a film of the abdomen to detect radiopaque paint chips or other leaded materials in the gastrointestinal tract and a film of the metaphyseal plate of a growing "long" bone, usually the proximal fibula, to detect interference with calcium deposition, the so-called "lead line."[42] Because this phenomenon is usually seen only after several weeks of increased lead absorption in children whose blood lead levels may exceed 50 μg/dL, its presence or absence may help to determine the duration of increased lead exposure.

Once a diagnosis of plumbism has been made, a child's condition and the effect of intervention should be monitored by serial venous determinations of the blood lead and, if available, erythrocyte protoporphyrin levels. If iron deficiency is present, iron studies should also be repeated periodically to monitor compliance with iron replacement therapy.

The lead mobilization test may be used to assess the "mobilizable" pool of lead in a child for whom chelation therapy is contemplated. Lead mobilization is determined by measuring lead diuresis in a timed urine collection following a single dose of chelating agent.[43,44] This test is most helpful in determining which children with blood lead concentrations in the range of 25 to 55 μg/dL will require a full course of chelation therapy and also in determining the advisability of further chelation in a child already receiving therapy. It should be noted that the erythrocyte protoporphyrin level is not a useful predictor of the amount of chelatable lead and may, in fact, be misleading in this regard.

Therapeutic modalities include removing the child from lead exposure, improving nutrition, administration of iron supplements, and chelation therapy.[40,45–47] In children with mild increased lead absorption, the efficacy of chelation therapy to modify neurobehavioral outcomes of lead toxicity is unproven; but, in children who have blood lead levels between 25 and 55 μg/dL and a positive lead mobilization test, it is highly desirable to rapidly decrease the readily mobile, potentially most toxic fraction of body lead stores by three to five days of $CaNa_2$-ethylenediaminetetraacetic acid (calcium disodium EDTA) therapy.[40]

Long-term follow-up is indicated in all cases of lead exposure. Children near or at school age who have a history of plumbism should have a neuro-

psychologic evaluation to identify potential learning handicaps, and school authorities should be encouraged to offer appropriate guidance.

RECOMMENDATIONS

Our recommendations rest on three premises: (1) that exposure to lead is widespread; (2) that lead causes neuropsychologic and other serious impairments in children at relatively low levels of exposure; and (3) that the neuropsychologic effects of lead, even in asymptomatic children, are largely irreversible. Guided by these premises, the goal of our recommendations is to prevent lead absorption.

Pediatricians must play a central role in this prevention. Although our recommendations are divided into two categories—those directed principally to practitioners and those directed to government agencies—the distinction is somewhat artificial. Throughout the past five decades, pediatricians, acting individually, as well as collectively through the Academy, have been prime movers in stimulating the agencies of government to protect the children of the United States against exposure to lead. It is important that this tradition of public involvement continue and that pediatricians continue to act publicly as advocates for the health of children.

Recommendations for Practitioners

1. The Academy recommends that the erythrocyte protoporphyrin test be used for screening children for lead toxicity, when that test is available. Additionally, the erythrocyte protoporphyrin test is a sensitive indicator of subclinical iron deficiency and may add complementary information to the determination of hematocrit values. It will not, however, identify children with anemia due to acute blood loss or hemoglobin C, SS, SC, or E disease. The Academy encourages clinical and hospital laboratories to make the erythrocyte protoporphyrin test widely and economically available.

2. Upon consideration of recent CDC recommendations, the Academy recommends that, ideally, all preschool children should be screened for lead absorption by means of the erythrocyte protoporphyrin test. However, it is recognized that the incidence of lead exposure may be so low in certain areas that pediatricians may prudently consider their patients to be at little risk of lead toxicity; therefore, the following priority guidelines ranked from highest to lowest are offered to assist pediatricians in deciding which children to screen. (a) children, 12 to 36 months of age, who live in or are frequent visitors in older, dilapidated housing (highest); (b) children, 9 months to 6 years of age, who are siblings, housemates, visitors, and playmates of children with known lead toxicity; (c) children of any age living in older housing where renovation is occurring; (d) children, 9 months to 6 years of age, living in older, dilapidated housing; (e) children, 9 months to 6 years of age, who live near lead smelters and processing plants or whose parents or other household members participate in a lead-related occupation or hobby. Frequency of screening should be flexible but should be guided by consideration of a child's age, nutrition and iron status, and housing age, housing condition, and population density. The first erythrocyte protoporphyrin test should generally occur at the same time as the determination of the hematocrit, which typically is performed between 9 and 15 months of age. Because the prevalence of lead poisoning increases sharply at 18 to 24 months of age, any child judged to be at elevated risk of plumbism should have a second erythrocyte protoporphyrin test performed at or about 18 months of age and at frequent intervals (3 to 6 months) thereafter appropriate to the degree of risk. Surveillance should continue routinely up to age 6 years and, if appropriate, longer.

3. The Academy recommends that any child, in whom increased lead absorption or lead poisoning has been confirmed by venous blood lead determination, be followed closely by means of repeat venous tests. For such children, abatement of environmental sources of lead is essential.

4. The Academy notes that some predisposing factors for lead poisoning, iron deficiency in particular, are preventable. Pediatricians should make vigorous efforts to identify and correct iron deficiency, calcium deficiency, and other nutritional deficiencies, particularly in children from areas of high lead exposure.

5. The Academy recommends that pediatricians attempt vigorously to educate parents, particularly parents of children in high-risk populations, about the hazards of lead, its sources and routes of absorption, and safe approaches to the prevention of exposure.

Recommendations for Public Agencies

1. The Academy recommends that reporting of cases of lead poisoning to state health departments be mandatory in all states.

2. The Academy notes that, in the present approach to screening for lead, inspection of a child's environment is generally undertaken only when an elevated blood lead level is found. In effect, children are used as biologic monitors for environmental lead. The Academy recommends that this sequence be reversed. A national program for systematic screening of lead hazards in housing is overdue. The enormity of the task favors a stepwise ap-

proach. Suggested approaches might include: screening of oldest housing, followed by newer housing; screening of housing in inner cities, then in less densely populated areas; and targeted screening of housing with small children.

3. The Academy supports the prompt, vigorous, and safe abatement of all environmental lead hazards. The US Department of Housing and Urban Development, state health departments, and local health departments should require that all hazardous lead-based paint (exterior and interior) be removed from all housing. Development of methods of abatement, which are safer and more effective than those currently in use (torches, heat guns, and sanders) must be given high priority to prevent the further endangering of lead-poisoning victims. The US Environmental Protection Agency is urged to persist in its laudable plan to promptly and finally remove all lead from gasoline.

4. The Academy urges the US Congress and the US Department of Health and Human Services to become fully cognizant of the high prevalence of childhood lead poisoning in the United States, its irreversible consequences, and its great human and fiscal costs. Restoration of funding is urgently needed for screening, hazard identification, and hazard abatement.

5. The Academy recommends that state health departments and Academy chapters exert their maximum influence to assure that state licensing agencies permit laboratories to perform blood lead and erythrocyte protoporphyrin tests only if those laboratories consistently meet criteria for accuracy and repeatability as determined by their performance in interlaboratory proficiency-testing programs.

SUMMARY

Patterns of childhood lead poisoning have changed substantially in the United States. The mean blood lead level has declined, and acute intoxication with encephalopathy has become uncommon. Nonetheless, between 1976 and 1980, 780,000 children, 1 to 6 years of age, had blood lead concentrations of 30 μg/L or above. These levels of absorption, previously thought to be safe, are now known to cause loss of neurologic and intellectual function, even in asymptomatic children. Because this loss is largely irreversible and cannot fully be restored by medical treatment, pediatricians' efforts must be directed toward prevention. Prevention is achieved by reducing children's exposure to lead and by early detection of increased absorption.

Childhood lead poisoning is now defined by the Academy as a whole blood lead concentration of 25 μg/L or more, together with an erythrocyte protoporphyrin level of 35 μg/dL or above. This definition does not require the presence of symptoms. It is identical with the new definition of the US Public Health Service. Lead poisoning in children previously was defined by a blood lead concentration of 30 μg/dL with an erythrocyte protoporphyrin level of 50 μg/dL.

To prevent lead exposure in children, the Academy urges public agencies to develop safe and effective methods for the removal and proper disposal of all lead-based paint from public and private housing. Also, the Academy urges the rapid and complete removal of all lead from gasoline.

To achieve early detection of lead poisoning, the Academy recommends that all children in the United States at risk of exposure to lead be screened for lead absorption at approximately 12 months of age by means of the erythrocyte protoporphyrin test, when that test is available. Furthermore, the Academy recommends follow-up erythrocyte protoporphyrin testing of children judged to be at high risk of lead absorption. Reporting of lead poisoning should be mandatory in all states.

ACKNOWLEDGMENT

We are grateful for the assistance of J. Julian Chisolm, Jr, MD, Jane L. Lin-Fu, MD, Vernon Houk, MD, John Stevenson, MD, and John F. Rosen, MD.

Gerald M. Breitzer, DO
Chuck Williams, MD

AAP Section Liaison
Jerry Foster, MD
Joyce A. Schild, MD

REFERENCES

1. Centers for Disease Control: Surveillance of childhood lead poisoning—United States. *MMWR* 1982;31:132–134
2. Mahaffey KR, Annest JL, Roberts J, et al: National estimates of blood lead levels: United States 1976–1980: Associated with selected demographic and socioeconomic factors. *N Engl J Med* 1982;307:573–579
3. Klein R: Lead poisoning. *Adv Pediatr* 1977;24:103–132
4. Annest JL, Pirkle JL, Makuc D, et al: Chronological trend in blood lead levels between 1976 and 1980. *N Engl J Med* 1983;308:1373–1377
5. Piomelli S, Corash L, Corash MB, et al: Blood lead concentrations in a remote Himalayan population. *Science* 1980; 210:1135–1137
6. Grandjean P, Fjerdingstad E, Nielsen OV: Lead concentration in mummified nubian brains. Presented at the International Conference on Heavy Metals in the Environment, Toronto, Canada, Oct 27–31, 1975
7. Hernberg S: Biochemical and clinical effects and responses as indicated by blood concentration, in Singhal RL, Thomas JS (eds): *Lead Toxicity*. Baltimore, Urban & Schwarzenberg, Inc, 1980
8. Berlin A, Schaller KH, Grimes H, et al: Environmental exposure to lead: Analytical and epidemiological investigations using the European standardized method for blood delta-aminolevulinic acid dehydratase activity determination. *Int Arch Occup Environ Health* 1977;39:135–141
9. Piomelli S, Seaman C, Zullow D, et al: Threshold for lead damage to heme synthesis in urban children. *Proc Natl Acad Sci USA* 1982;79:3335–3339
10. Mahaffey KR, Rosen JF, Chesney RW, et al: Association between age, blood lead concentration, and serum 1,25-dihydroxycalciferol levels in children. *Am J Clin Nutr* 1982;35:1327–1331
11. Rosen JF, Chesney RW, Hamstra A, et al: Reduction in 1,25-dihydroxyvitamin D in children with increased lead absorption. *N Engl J Med* 1980;302:1128–1131
12. Needleman H, Gunnoe C, Leviton A, et al: Deficits in psychologic and classroom performance of children with elevated dentine lead levels. *N Engl J Med* 1979;300:689–695
13. Winneke G, Kramer G, Brockhaus A, et al: Neuropsychological studies in children with elevated tooth lead concentration. *Int Arch Occup Environ Health* 1982;51:169–183
14. Yule W, Lansdowne R, Millar IB, et al: The relationship between blood lead concentrations, intelligence and attainment in a school population: A pilot study. *Dev Med Child Neurol* 1981;23:567–576
15. Rutter M: Low level lead exposure: Sources, effects, and implications, in Rutter M, Jones RR, (eds): *Lead vs. Health*. New York; John Wiley & Sons, 1983, pp 333–370
16. Schwartz J, Angle C, Pitcher H: Relationship between childhood blood lead levels and stature. *Pediatrics* 1986;77:281–288
17. Centers for Disease Control: Preventing lead poisoning in young children. *MMWR* 1985;34:67–68
18. Landrigan PJ, Gehlbach SH, Rosenblum BF, et al: Epidemic lead absorption near an ore smelter: The role of particulate lead. *N Engl J Med* 1975;292:123–129
19. *Assessment of the Safety of Lead and Lead Salts in Food: A Report of the Nutrition Foundation's Expert Advisory Committee*. Washington, DC, The Nutrition Foundation, 1982
20. Bureau of the Census. *Statistical Abstracts of the United States, 1980*, ed 101. Washington, DC, US Department of Commerce, 1980
21. Bureau of the Census: *Annual Housing Survey, 1979*, Current Housing reports series H-150-79. Washington DC, US Department of Commerce, 1979
22. Fischbein A, Anderson KE, Sassa S, et al: Lead poisoning from "do-it-yourself" heat guns for removing lead-based paint: Report of two cases. *Environ Res* 1981;24:425–431
23. Landrigan PJ, Baker EL, Himmelstein JS, et al: Exposure to lead from the Mystic river bridge: The dilemma of deleading. *N Engl J Med* 1982;306:673–676
24. Bose A, Vashistha K, O'Loughlin BJ: *Azarcón por Empacho*—Another cause of lead toxicity. *Pediatrics* 1983;72:106–108
25. Centers for Disease Control: Folk remedy-associated lead poisoning in Hmong children—Minnesota. *MMWR* 1983; 32:555–556
26. Ali AR, Smalls ORC, Aslam M: Surma and lead poisoning. *Br Med J* 1978;3:915–916
27. Shaltout A, Yaish SA, Fernando N: Lead encephalopathy in infants in Kuwait. *Ann Trop Paediatr* 1981;1:209–215
28. Miller C: The pottery and plumbism puzzle. *Med J Austr* 1982;30:442–443
29. Osterud HT, Tufts E, Holmes MS: Plumbism at the Green Parrot goat farm. *Clin Toxicol* 1973;6:1–7
30. Baker EL Jr, Folland DS, Taylor TA, et al: Lead poisoning in lead workers and their children: Home contamination with industrial dust. *N Engl J Med* 1977;296:260–261
31. Chisolm JJ Jr: Fouling one's own nest. *Pediatrics* 1978;62:614–617
32. Ziegler EE, Edwards BB, Jansen RL, et al: Absorption and retention of lead by infants. *Pediatr Res* 1978;12:29–34
33. Roels HA, Buchet JP, Lauwerys RR, et al: Exposure to lead by the oral and the pulmonary routes of children living in the vicinity of a primary lead smelter. *Environ Res* 1980;22:81–94
34. Sayre JW, Charney E, Vostal J, et al: House and hand dust as a potential source of childhood lead exposure. *Am J Dis Child* 1974;127:167–171
35. Charney E, Kessler B, Farfel M, et al: Childhood lead poisoning: A controlled trial of the effect of dust-control measures on blood lead levels. *N Engl J Med* 1983;309:1089–1093
36. Barltrop D: The prevalence of pica. *Am J Dis Child* 1966;112:116–123
37. Mahaffey KR: Nutritional factors in lead poisoning. *Nutr Rev* 1981;39:353–362
38. Nathan D, Oski F: *Hematology of Infancy and Childhood*, ed 2. Philadelphia, WB Saunders Co, 1981
39. Yip R, Schwartz S, Deinard AS: Screening for iron deficiency with the erythrocyte protoporphyrin test. *Pediatrics* 1983;72:214–219
40. Piomelli S, Rosen JF, Chisolm JJ Jr, et al: Management of childhood lead poisoning. *J Pediatr* 1984;105:523–532
41. *Preventing Lead Poisoning in Young Children*, publication No. 00-2629. Atlanta, Centers for Disease Control, April 1978
42. Blickman JH, Wilinson R, Graef J: The lead line revisited. *AJR* 1986;74:89–93
43. Markowitz ME, Rosen JF: Assessment of lead stores in children: Validation of 8-hour $CaNa^2EDTA$ provocative test. *J Pediatr* 1984;104:337–341
44. Teisinger J, Srbova J: The value of mobilization of lead by calcium ethylene diaminetetraacetate in the diagnosis of lead poisoning. *Br J Ind Med* 1959;16:148–153
45. Chisolm JJ Jr, Barltrop D: Recognition and management of children with increased lead absorption. *Arch Dis Child* 1979;54:249–262
46. Graef JW: Clinical outpatient management of childhood lead poisoning, in Chisolm JJ Jr, O'Hara D (eds): *Lead Absorption in Children*. Baltimore, Urban & Schwarzenberg, 1982, pp 153–164
47. Chisolm JJ Jr: Management of increased lead absorption—Illustrative cases, in Chisolm JJ Jr, O'Hara D (eds): *Lead Absorption in Children*. Baltimore, Urban & Schwarzenberg, 1982, pp 172–182